

# Assessing Volume Status



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## KEYWORDS

- Shock • Fluid resuscitation • Cardiac output • Stroke volume • Hemorrhage
- Hemodynamic monitoring • Noninvasive monitoring • Critical care

## KEY POINTS

- Shock is a physiologic state associated with high morbidity and mortality rates.
- Fluid resuscitation has long been a part of the acute resuscitation armamentarium.
- Emergency physicians have a major impact on patient survival. The clinician has several tools available to evaluate volume status.
- Each modality has its benefits and limitations, but, to date, no one test can indicate with 100% accuracy which patients will be truly volume responsive.

## INTRODUCTION

Shock, by definition, is a condition of inadequate tissue perfusion; during resuscitation, the clinician's goal is to restore the patient's perfusion of organs and tissues. The importance of early goal-directed therapy and early intervention by emergency physicians for hypotensive patients has been shown in multiple studies,<sup>1,2</sup> most classically by Rivers and colleagues<sup>3</sup> for patients presenting in septic shock. One of the most important principles of Rivers' study was the use of targeted and aggressive fluid resuscitation to improve tissue perfusion.

Fluid resuscitation has long been a part of the acute resuscitation armamentarium. Its goal is to increase cardiac filling and stroke volume (ie, cardiac output). What is often less clear, however, is accurately determining whether the patient who is currently hypotensive will actually respond to a fluid bolus (**Fig. 1**); that is, before administering a fluid bolus, it is very difficult to determine if the patient will respond.

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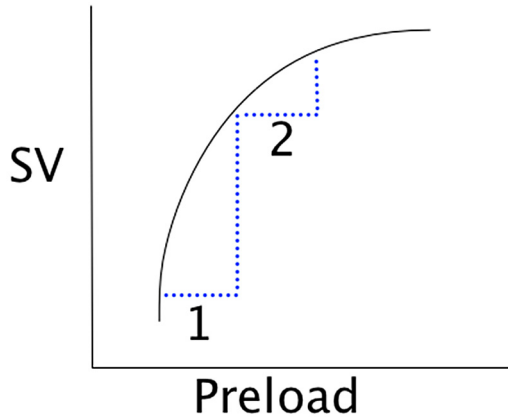
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**Fig. 1.** Frank-Starling relationship of the heart. When the left ventricle is underfilled (1), an increase in preload brings on a large increase in stroke volume, and the patient is said to be volume responsive. When the left ventricle is closer to maximum end-diastolic volume (2), the increase in stroke volume is minimal for the same increase in preload.

This goal has proved to be perpetually elusive. In most studies, more than 40% of intensive care unit patients receiving a fluid bolus did not show the desired increase in cardiac output.<sup>4</sup> The harm of unnecessary and excessive fluid administration was shown in multiple studies, which suggest that the deleterious effects of fluid overload likely go far beyond simple pulmonary edema.<sup>5–9</sup> The endpoint of a goal-directed fluid strategy has placed increasing emphasis on finding a true “Goldilocks” state of fluid balance (ie, to not administer too much fluid or withhold too much).

This article describes the various tools available to the emergency physician to help answer an often perplexing question at the bedside: “Does this hemodynamically unstable patient need intravenous fluids?”

### PHYSICAL EXAMINATION

Classic medical school teaching tells us to rely on the physical examination and vital signs to guide our resuscitation strategy, and this instruction extends into our clinical practice.<sup>10</sup> However, when the objective findings are evaluated for their correlation with actual changes in perfusion and cardiac output, few of them perform well.<sup>11,12</sup>

One example is estimation of jugular venous pressure during the physical examination, which is used to evaluate abnormalities of the left-sided circulation. Not only is jugular venous pressure difficult to measure under some circumstances (eg, when assessing an obese patient), but its true value can be confounded by numerous coexisting pathologies (eg, valvular disease, pulmonary hypertension). Another commonly used marker, urine output, can fail to accurately indicate the success of resuscitative efforts because it can be difficult to distinguish oliguria secondary to circulatory dysfunction (prerenal azotemia) from intrinsic kidney injury (eg, acute tubular necrosis) in the acute care setting. Even our most common and fundamental goal in resuscitation—improvement in the arterial pressure—does not necessarily correlate with improved cardiac output and, therefore, does not necessarily reflect improved perfusion.<sup>13</sup>

Once the limitations of the physical examination were realized, advanced resuscitation techniques for patients in shock shifted to using cardiac filling pressures as a

guide to fluid therapy. The use of filling pressures makes theoretic sense because, as indicated by physiology and the Frank-Starling curve (see [Fig. 1](#)), ventricular pressures are directly proportional to ventricular volume until they reach a certain point at which this relationship is lost. Therefore, the goal during resuscitation is to administer intravenous fluids to optimally fill the left ventricle, which will increase the stroke volume, but not overfill it, which could result in deleterious side effects such as pulmonary edema.

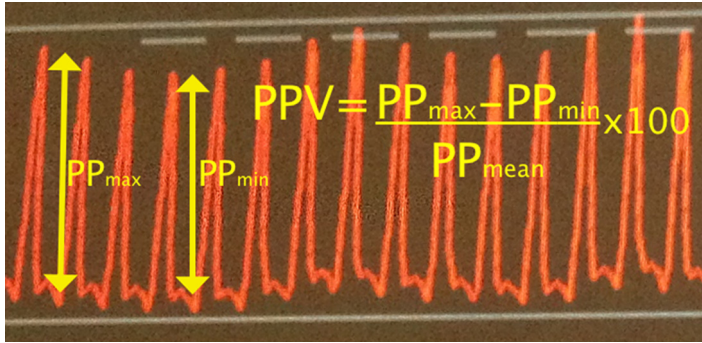
When these filling pressures are interpreted randomly, they are known as *static measurements* and are thought to be a direct reflection of the ventricular pressure and hence the volume of the cardiac chambers. Central venous pressure (CVP) is the most popular of the static pressures and continues to be recommended in resuscitative protocols and guidelines.<sup>3,10</sup> Despite these recommendations, the reliability of CVP for guiding fluid resuscitation is controversial because of variations in patients' physiologies (eg, valvular pathology, right ventricular dysfunction)<sup>14</sup> as well as persistent inaccuracy when it is used as a predictor of response to a volume bolus.<sup>15</sup> An additional limitation to use of the CVP is that it requires insertion of a central venous catheter, which is invasive, and its placement is not readily possible in all emergency departments.

Another example of a static measurement of volume status is the estimation of right atrial pressure, based on an ultrasound image of the absolute size of the inferior vena cava (IVC) and its measured respiratory variation.<sup>16</sup> The IVC can be used to estimate right atrial pressures and, by indirect correlation, the CVP, which is the specified target in early goal-directed therapy. Ultrasound scan is an attractive hemodynamic monitor because it is noninvasive, rapid, and reproducible. Although there is debate as to its accuracy when predicting fluid responsiveness, it can be a useful measure when used as part of a comprehensive resuscitative protocol.<sup>17,18</sup>

The counterpart to static measurement of volume status is dynamic measurement. Dynamic measurements differ from static measurements because they involve the monitoring of hemodynamics followed by an intervention (eg, fluid bolus). Although dynamic measurements are better than static markers in predicting volume responsiveness,<sup>4,19,20</sup> their routine use in the emergency department has 2 major limitations. First, dynamic measurements require the concurrent use of monitoring devices that can detect increases in stroke volume (eg, ultrasound scan) during the assessment (such devices are explained in further detail later in this article). Second, most dynamic assessments require that patients be intubated and mechanically ventilated. This method is chosen because mechanical ventilation increases intrathoracic pressure, producing a significant respirophasic variation in preload, which mimics a fluid bolus, with venous return being at a minimum at end-inspiration (highest intrathoracic pressure) and a maximum at end-expiration (lowest intrathoracic pressure).

## **PULSE PRESSURE VARIATION AND STROKE VOLUME VARIATION**

Pulse pressure variation (PPV), a well-studied dynamic marker of cardiac output, measures the change in pulse pressure (PP) throughout a respiratory cycle on a mechanical ventilator ([Fig. 2](#)). An arterial line is required to measure PPV, because systolic and diastolic measurements must be taken at specific points in the respiratory cycle. Once these measurements are recorded, the PPV can be calculated. Specialized monitors automatically perform both the measurements and the calculations in real time. Systolic pressure variation uses the same principal as PPV, except only the systolic pressures are measured at specific times in the respiratory cycle; therefore, it is not as reliable as PPV.



**Fig. 2.** Calculation of PPV using the tracing from an arterial catheter monitor. PPmax, maximum pulse pressure; PPmean, mean pulse pressure; PPmin, minimum pulse pressure (calculated by device).

PPV is a well-validated predictor of fluid responsiveness and can be applied in variety of clinical settings (emergency department, intensive care unit, operating room).<sup>21–23</sup> A PPV greater than 12.5% suggests that a patient’s cardiac output would increase when given a fluid bolus. Conversely, a PPV less than 12.5% suggests that giving a fluid bolus would neither increase cardiac output nor improve hemodynamic status.<sup>24</sup>

The use of PPV for volume responsiveness in the emergency department has several limitations (**Box 1**). One limitation is that this measurement reflects a patient’s hemodynamic status at only one point in time, so PPV must be calculated several times during resuscitation, especially before administering a fluid bolus.

### IVC DISTENSIBILITY INDEX

The IVC distensibility index (IVCdi) is another commonly used dynamic assessment to determine fluid responsiveness. Ultrasound scan is used to assess the change in size of the IVC during a respiratory cycle in a mechanically ventilated patient. As was the case for PPV, positive intrathoracic pressure is essential to use this technique accurately. The benefit of IVCdi is that it is noninvasive (no arterial line is required) and is reproducible multiple times at the bedside.

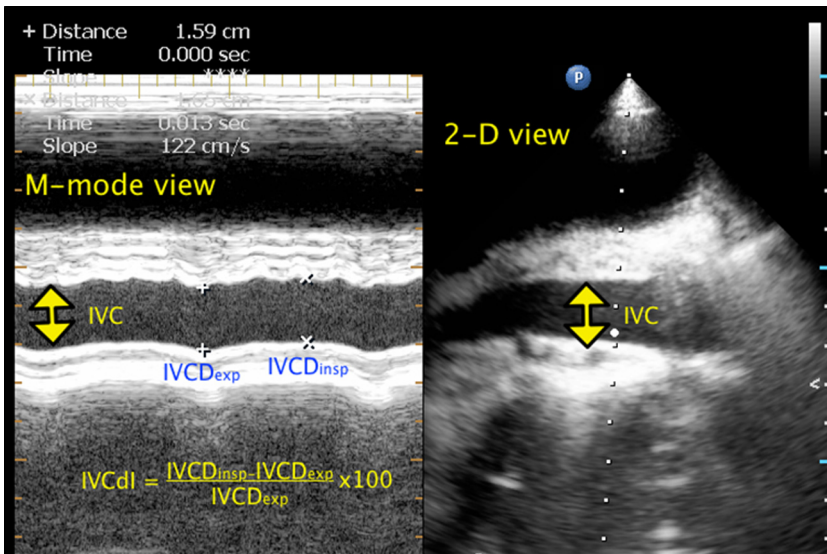
During the delivery of a mechanical breath, a patient’s intrathoracic pressure increases, and venous return decreases, resulting in IVC dilation (an increase in the diameter of the vessel). When the mechanical breath is released, the intrathoracic pressure decreases, and the IVC returns to its normal diameter. The degree of IVC diameter change is measured with ultrasound scan and then the IVCdi is calculated (**Fig. 3**).

#### Box 1

##### Limitations of dynamic indices

For IVCdi, PPV, or SVV to accurately predict volume responsiveness, the following criteria must be met:

- Mechanical ventilation
- Tidal volume of at least 8 mL/kg of ideal body weight
- Initiating no spontaneous breaths
- No cardiac arrhythmias



**Fig. 3.** IVC distensibility index. Calculation of IVCdI using M-mode ultrasound scan. IVCDexp, IVC diameter at end-expiration; IVCDinsp, IVC diameter at end-inspiration.

Many findings suggest that IVCdI is a useful bedside tool for assessing the status of the intravascular volume for patients undergoing fluid resuscitation. There has been, however, variation in the agreed cutoff values of the IVCdI across studies. Some studies found a cutoff of 12%, but others proposed values as high as 36%.<sup>25–27</sup>

Limitations of measuring the IVCdI are similar to those described for PPV (see **Box 1**). There is also debate in the literature on what level of the IVC is the best site for measurement. The location of measurement (eg, at the level of the hepatic or renal vein) can affect the recorded value of the IVC diameter.<sup>28</sup>

### END-EXPIRATORY OCCLUSION TEST

The end-expiratory occlusion (EEO) test is a relatively new technique for dynamic assessment of fluid responsiveness in mechanically ventilated patients. The EEO test is executed by placing a 15-second expiratory hold on the ventilator (at the end of expiration). A prolonged state of increased preload is created by holding the positive pressure ventilation at its lowest pressure, mimicking a fluid challenge. This test has the advantage of being able to assess fluid responsiveness even in the presence of arrhythmias, a notable difference from the techniques previously discussed.<sup>29–31</sup>

This technique requires concurrent measurement of stroke volume or cardiac output. Although this is a notable limitation, several monitoring approaches have been reported, such as measuring cardiac output by thermodilution or waveform analysis. When such monitoring is available, if the stroke volume or cardiac output during the final 5 seconds of the expiratory hold is 5% higher than the premaneuver value, the patient will likely respond to the administration of a fluid bolus.<sup>29,31</sup>

### PASSIVE LEG RAISE

The dynamic techniques described above have one thing in common: the patient must be mechanically ventilated. Although these dynamic indices are fairly well validated, it

would be helpful to have a test that is valid for spontaneously breathing patients. The passive leg raise (PLR) is the only dynamic index to date that has the ability to predict the response to fluids in a spontaneously breathing patient (Fig. 4).<sup>32,33</sup>

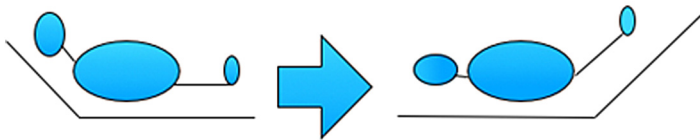
The PLR technique mobilizes pooled venous blood in the lower extremities (approximately 150–300 mL<sup>34</sup>) to the central circulation as an autologous and reversible fluid bolus. In this way, extrinsic fluids are not necessary, and patients do not receive an unnecessary fluid bolus if they are not volume responsive. The PLR is performed by first measuring the baseline stroke volume or cardiac output, followed by remeasuring it 30 to 90 seconds after lifting the patient's lower extremities.<sup>35</sup> A 10% to 15% change in stroke volume or cardiac output is an indicator that the patient will respond to exogenous fluid administration. Another benefit to this test is that it has been validated for patients with cardiac arrhythmias (eg, atrial fibrillation).<sup>36</sup> Similar to the EEO test, studies have used several monitoring systems, including bedside echocardiography, arterial waveform analysis, and bioactance, to determine change in cardiac output or stroke volume.<sup>37</sup>

### DEVICES FOR HEMODYNAMIC MONITORING

The methods described in this article permit the clinician to deliver fluid boluses in a goal-directed manner. Unfortunately, many of these methods are labor intensive and require the clinician to return to the bedside repeatedly to assess the patient's volume status. This requirement can be challenging and impractical in a busy emergency department.

Devices that automatically monitor and display a patient's hemodynamic information are desirable because they allow the use of nurse-directed resuscitation protocols while the clinician leaves the bedside temporarily. Another benefit of such monitors is as an early warning system that alerts clinicians to changes in a patient's hemodynamic status. These devices display hemodynamic information such as stroke volume, cardiac output, and stroke volume variation (SVV).

SVV is the amount of variation that occurs over a respiratory cycle in mechanically ventilated patients. It is similar in concept to PPV, except that a device is used to estimate the actual stroke volume rather than using the pulse pressure as a surrogate. Similar to PPV, SVV has been validated across multiple situations as a predictor of fluid responsiveness.<sup>38,39</sup> An SVV measurement greater than 11.5% suggests that giving a fluid bolus would be beneficial.<sup>24</sup> Conversely, an SVV less than 11.5% suggests that a fluid bolus would not improve the patient's hemodynamics and might cause harm (eg, pulmonary edema). Although PPV and SVV are very similar, PPV seems to slightly outperform SVV<sup>23,24,38</sup> and requires the same conditions for validity as do PPV and IVCdI (see Box 1).



**Fig. 4.** Passive leg raising maneuver. The process begins with the head of the bed and the patient's trunk elevated to approximately 45°, at which time baseline parameters are recorded. The patient is then quickly laid back, with head and trunk flat and legs raised to 45°, allowing the venous blood pooled in the legs to return to the heart quickly, creating an autologous transfusion. Parameters should be rerecorded 30 to 90 seconds after raising the legs to evaluate for volume responsiveness.

Two main categories of devices provide hemodynamic data: invasive and noninvasive monitors. Each type has benefits and disadvantages, and neither is perfect. The choice of device depends on several factors, a full discussion of which can be found elsewhere.<sup>40</sup>

### INVASIVE HEMODYNAMIC MONITORS

The pulmonary artery catheter (PAC) measures stroke volume and cardiac output by measuring the amount of time it takes for a known quantity of fluid to traverse the right side of the heart. The longer it takes, the worse the stroke volume. One of the major criticisms of the PAC is its invasive nature, given that it must be inserted through the superior vena cava, into the right side of the heart, across the tricuspid and pulmonary valves, and into the pulmonary artery. Major complications associated with the PAC include pneumothorax, arterial puncture, and hematoma—complications associated with the placement of any central line. Complications specific to PAC include ventricular arrhythmias, ventricular rupture, and pulmonary artery rupture.<sup>41</sup>

Two commercially available devices have monitoring principles similar to the PAC but are less invasive: the PiCCO and LidCO systems. These devices use an arterial catheter to perform pulse-contour analysis, which is a technique to determine hemodynamic parameters from an arterial waveform.

Both devices are periodically calibrated to improve accuracy. Calibration is performed by injecting a fluid into a venous line (ice-cold saline for the PiCCO and lithium for LidCO) and measuring the fluid in the arterial circulation after it traverses the heart.<sup>42,43</sup> The PiCCO system measures the degree of temperature elevation of the saline injected, and the LidCO system measures the dilution of the lithium that was injected; these measurements are made via a sensor in an arterial line.

Monitoring stroke volume and cardiac output with these monitors helps the clinician determine whether a hemodynamically unstable patient would benefit from a fluid bolus, because the response to fluid bolus can be measured. For example, an increase in either parameter by 10% to 15% after a fluid bolus suggests the patient was volume responsive as part of the hemodynamic resuscitation.<sup>44</sup> However, failure to increase the stroke volume or cardiac output by 10% to 15% after a fluid bolus suggests that the hemodynamically unstable patient would not improve with fluid administration and should perhaps receive vasopressors or an inotrope.

The PiCCO device is considered a minimally invasive device because it requires the placement of a central venous catheter (the tip of which resides at the junction of the superior vena cava and the right atrium) and a femoral arterial line that will detect changes in saline temperature distally. The limitations of the PiCCO device include decreased accuracy in patients with low-flow states (eg, severe heart failure), valvular regurgitation, intracardiac shunts, and aortic aneurysms.<sup>43</sup>

The LidCO device is somewhat less invasive than the PiCCO in that it requires placement of only a peripheral intravenous line and a radial arterial line (a central venous catheter can be used if it is already available). The major limitations to the accuracy of the LidCO device are similar to those associated with the PiCCO device, including low flow states, valvular regurgitation, and intracardiac shunts. A limitation specific to the LidCO device is its use of lithium dye. Although this dye is safe for most patients, it should be avoided in certain situations, including patients who weigh less than 40 kg, pregnant patients, and those taking lithium as a medication.<sup>45</sup>

A third minimally invasive system is the Flo Trac/Vigileo system. This system differs from the monitors discussed above in that it requires placement of only an arterial catheter. The monitor displays hemodynamic data such as stroke volume, cardiac



output, and stroke volume variation by analyzing arterial waveforms and pressures and integrating this information into a complex algorithm based on the patient's demographics (height, sex, weight). This device does not require external calibration, and its manufacturer claims that the system is continuously fine tuning and auto calibrating each minute. Auto calibration is based on cadaveric studies, and its accuracy has been questioned.<sup>46</sup> The device has also been found to be inaccurate in certain clinical conditions (eg, in patients with rapidly changing cardiac output or high cardiac output states).<sup>47</sup>

Several studies found these minimally invasive monitors to be accurate. No single device, however, has gained widespread acceptance in emergency medicine; one of the major reasons for this is their invasiveness, requiring at least an arterial line (and a central line in the case of the PICCO). Despite their limitations, these devices provide clinicians with hemodynamic monitoring that is otherwise difficult to achieve with traditional methods.

### NONINVASIVE HEMODYNAMIC MONITORS

Invasive catheters have the potential to create complications, such as discomfort during placement, infection, and bleeding, so a noninvasive hemodynamic monitor is an appealing concept in the emergency department. Two examples of completely noninvasive monitors are the ultrasonic cardiac output monitor (USCOM) and the noninvasive cardiac output monitor (NICOM).

#### *USCOM*

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The USCOM is a small probe that can be applied intermittently to a patient's chest or suprasternal notch whenever hemodynamic information is needed. The device works by providing a continuous Doppler signal, which measures the velocity of blood flow in the aorta (parasternal view) or the pulmonary blood flow (suprasternal notch).<sup>43,48,49</sup> This information is used in conjunction with the patient's height to calculate several hemodynamic parameters, including stroke volume and cardiac output. Beyond the noninvasive nature of this device, another advantage is that bedside nurses can obtain the information for use in resuscitative algorithms.

The USCOM seems to be a promising addition to the emergency department armamentarium, although there are several limitations to its general use. The device can easily be applied to a patient, but obtaining an adequate and accurate Doppler signal can be a challenge for novice users and altogether impossible in a percentage of certain populations (eg, the elderly).<sup>50</sup> Another limitation is that hemodynamic information is not obtained continuously but rather only when the device is applied; this means that crucial information might be missed in patients with a rapidly changing hemodynamic state. Another criticism of the USCOM is the assumption that 2 patients with the same height will have the same hemodynamic changes when faced with a similar stressor, which might not actually be true. A detailed discussion of this concept is beyond the scope of this article, but it is a valid limitation that should be considered.<sup>51</sup>

#### *NiCOM: Bioreactance*

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The NiCOM requires the placement of 4 adhesive electrodes on the patient's anterior thorax. These electrodes measure changes in the frequency of electrical currents as they cross the thorax, converting this information into relevant hemodynamic information, including stroke volume, cardiac output, and stroke volume variation.<sup>52-54</sup> Newer versions of this device incorporate the use of a passive leg raise maneuver to determine volume responsiveness.<sup>55,56</sup> This device has been found to correlate well with



more invasive means of hemodynamic monitoring, such as the pulmonary artery catheter.

The greatest clinical benefit of this device is that it provides continuous hemodynamic monitoring noninvasively for critically ill patients. Rapidly changing hemodynamics are more likely to be detected with this monitor compared with monitors that use intermittent measurements.<sup>54</sup> Additionally, little training is required to set up and use the device because the sensors are applied to the thorax and do not require constant manipulation, minimizing sampling errors between readings. More clinical trials are needed to determine whether this device improves patient outcomes, but it is a promising technology for continuous hemodynamic monitoring in the emergency department.

## SUMMARY

Shock is a physiologic state associated with high morbidity and mortality rates. Emergency physicians have a major impact on patient survival. The clinician has several tools available to evaluate volume status. Each modality has its benefits and limitations but, to date, no one test can indicate with 100% accuracy which patients will be truly volume responsive. Although the search for the Holy Grail of a perfect intravascular monitor continues, we must remember the importance of early, aggressive, and goal-directed interventions for patients in shock. We, therefore, must be astute clinicians and integrate the multitude of variables available at the bedside to assist in our decision making; this is truly the art of medicine. Finally, there is no substitute for the most important intervention—the frequent presence of the physician at the patient's bedside.

## REFERENCES

1. Kumar A, Roberts D, Wood KE, et al. Duration of hypotension before initiation of effective antimicrobial therapy is the critical determinant of survival in human septic shock. *Crit Care Med* 2006;34:1589–96.
2. Marchick MR, Kline JA, Jones AE. The significance of non-sustained hypotension in emergency department patients with sepsis. *Intensive Care Med* 2009;35:1261–4.
3. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368–77.
4. Michard F, Teboul J. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest* 2002;121:2000–8.
5. Vincent JL, Sakr Y, Ranieri VM, et al. Sepsis in European intensive care units: results of the SOAP study. *Crit Care Med* 2006;34:344–53.
6. Rosenberg AL, Dechert RE, Park PK, et al. Association of cumulative fluid balance on outcome in acute lung injury: a retrospective review of the ARDSnet tidal volume study cohort. *J Intensive Care Med* 2009;24:35–46.
7. Boyd JH, Forbes J, Nakada TA, et al. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. *Crit Care Med* 2011;39:259–65.
8. Sadaka F, Juarez M, Naydenov S, et al. Fluid resuscitation in septic shock: the effect of increasing fluid balance on mortality. *J Intensive Care Med* 2013;29:213–7.
9. Micek ST, McEvoy C, McKenzie M, et al. Fluid balance and cardiac function in septic shock as predictors of hospital mortality. *Crit Care* 2013;17:R246.

10. Dellinger RP, Levy MM, Rhodes A, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock, 2012. *Intensive Care Med* 2013;39:165–228.
11. Rady MY, Rivers EP, Martin GB, et al. Continuous central venous oximetry and shock index in the emergency department: use in the evaluation of clinical shock. *Am J Emerg Med* 1992;10:538–41.
12. Rady MY, Rivers EP, Nowak RM. Resuscitation of the critically ill in the ED: responses of blood pressure, heart rate, shock index, central venous oxygen saturation, and lactate. *Am J Emerg Med* 1996;14:218–25.
13. Pierrakos C, Velissaris D, Scolletta S, et al. Can changes in arterial pressure be used to detect changes in cardiac index during fluid challenge in patients with septic shock? *Intensive Care Med* 2012;38:422–8.
14. Nahouraii RA, Rowell SE. Static measures of preload assessment. *Crit Care Clin* 2010;26:295–305.
15. Marik P, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. *Chest* 2008;134:172–8.
16. Otto C. *Textbook of clinical echocardiography*. 2nd edition. Philadelphia: WB Saunders; 2000.
17. Perera P, Mailhot T, Riley D, et al. The RUSH exam: rapid ultrasound in shock in the evaluation of the critically ill. *Emerg Med Clin North Am* 2010;28:29–56.
18. Ferrada P, Anand RJ, Whelan J, et al. Qualitative assessment of the inferior vena cava: useful tool for the evaluation of fluid status in critically ill patients. *Am Surg* 2012;78:468–70.
19. Michard F, Boussat S, Chemla D, et al. Relationship between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. *Am J Respir Crit Care Med* 2000;162:134–8.
20. Osman D, Ridel C, Ray P, et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. *Crit Care Med* 2007;35:64–8.
21. Solus-Biguenet H, Fleyfel M, Tavernier B, et al. Non-invasive prediction of fluid responsiveness during major hepatic surgery. *Br J Anaesth* 2006;97:808–16.
22. Zhang Z, Lu B, Sheng X, et al. Accuracy of stroke volume variation in predicting fluid responsiveness: a systematic review and meta-analysis. *J Anesth* 2011;25: 904–16.
23. Biais M, Cottenceau V, Stecken L, et al. Evaluation of stroke volume variations obtained with the pressure recording analytic method. *Crit Care Med* 2012;40: 1186–91.
24. Marik PE, Cavallazzi R, Vasu T, et al. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med* 2009;37:2642–7.
25. Feissel M, Michard F, Faller J, et al. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med* 2004;30:1834–7.
26. Barbier C, Loubieres Y, Schmit C, et al. Respiratory changes in inferior vena cava diameter are helpful in predicting fluid responsiveness in ventilated septic patients. *Intensive Care Med* 2004;30:1740–6.
27. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med* 2004;30:1734–9.
28. Machare-Delgado E, Decaro M, Marik PE. Inferior vena cava variation compared to pulse contour analysis as predictors of fluid responsiveness: a prospective cohort study. *J Intensive Care Med* 2011;26:116–24.

29. Wallace DJ, Allison M, Stone MB. Inferior vena cava collapse during respiration is affected by the sampling location: an ultrasound study in healthy volunteers. *Acad Emerg Med* 2010;17:96–9.
30. Monnet X, Osman D, Ridet C, et al. Predicting volume responsiveness by using the end-expiratory occlusion in mechanically ventilated intensive care unit patients. *Crit Care Med* 2009;37:951–6.
31. Monnet X, Bleibtreu A, Ferre A, et al. Passive leg-raising and end-expiratory occlusion tests perform better than pulse pressure variation in patients with low respiratory system compliance. *Crit Care Med* 2012;40:152–7.
32. Monnet X, Dres M, Ferre A, et al. Prediction of fluid responsiveness by a continuous non-invasive assessment of arterial pressure in critically ill patients: comparison with four other dynamic indices. *Br J Anaesth* 2012;109:330–8.
33. Teboul J, Monnet X. Prediction of volume responsiveness in critically ill patients with spontaneous breathing activity. *Curr Opin Crit Care* 2008;14:334–9.
34. Biais M, Vidil L, Sarrabay P, et al. Changes in stroke volume induced by passive leg raising in spontaneously breathing patients: comparison between echocardiography and Vigileo/FloTrac device. *Crit Care* 2009;13:R195.
35. Rutlen DL, Wackers FJ, Zaret BL. Radionuclide assessment of peripheral intravascular capacity: a technique to measure intravascular volume changes in the capacitance circulation in man. *Circulation* 1981;64:146–52.
36. Monnet X, Teboul J. Passive leg raising. *Intensive Care Med* 2008;34:659–63.
37. Cavallaro F, Sandroni C, Marano C, et al. Diagnostic accuracy of passive leg raising for prediction of fluid responsiveness in adults: systematic review and meta-analysis of clinical studies. *Intensive Care Med* 2010;36:1475–83.
38. Marik PE, Levitov A, Young A, et al. The use of bioactance and carotid Doppler to determine volume responsiveness and blood flow redistribution following passive leg raising in hemodynamically unstable patients. *Chest* 2013;143:364–70.
39. Cecconi M, Monti G, Hamilton MA, et al. Efficacy of functional hemodynamic parameters in predicting fluid responsiveness with pulse power analysis in surgical patients. *Minerva Anestesiol* 2012;78:527–33.
40. Reuter DA, Kirchner A, Felbinger TW, et al. Usefulness of left ventricular stroke volume variation to assess fluid responsiveness in patients with reduced cardiac function. *Crit Care Med* 2003;31:1399–404.
41. Vincent JL, Rhodes A, Perel A, et al. Clinical review: update on hemodynamic monitoring - a consensus of 16. *Crit Care* 2011;15:229.
42. Marik PE. Obituary: pulmonary artery catheter 1970 to 2013. *Ann Intensive Care* 2013;3:38.
43. Ramsingh D, Alexander B, Cannesson M. Clinical review: does it matter which hemodynamic monitoring system is used? *Crit Care* 2013;17(2):208.
44. Mohammed I, Phillips C. Techniques for determining cardiac output in the intensive care unit. *Crit Care Clin* 2010;26:355–64.
45. Napoli A. Physiologic and clinical principles behind noninvasive resuscitation techniques and cardiac output monitoring. *Cardiol Res Pract* 2012;2012:531908.
46. Reuter DA, Huang C, Edrich T, et al. Cardiac output monitoring using indicator-dilution techniques: basics, limits, and perspectives. *Anesth Analg* 2010;110:799–811.
47. Benington S, Ferris P, Nirmalan M. Emerging trends in minimally invasive haemodynamic monitoring and optimization of fluid therapy. *Eur J Anaesthesiol* 2009;26:893–905.

48. Mayer J, Boldt J, Poland R, et al. Continuous arterial pressure waveform-based cardiac output using the Flo Trac/Vigileo: a review and meta-analysis. *J Cardiothorac Vasc Anesth* 2009;23:401–6.
49. Boyle M, Steel L, Flynn GM, et al. Assessment of the clinical utility of an ultrasonic monitor of cardiac output (the USCOM) and agreement with thermodilution measurement. *Crit Care Resusc* 2009;11:198–203.
50. Tan HL, Pinder M, Parsons R, et al. Clinical evaluation of USCOM ultrasonic cardiac output monitor in cardiac surgical patients in intensive care unit. *Br J Anaesth* 2005;94:287–91.
51. Chong SW, Peyton PJ. A meta-analysis of the accuracy and precision of the ultrasonic cardiac output monitor (USCOM). *Anaesthesia* 2012;67:1266–71.
52. Huang L, Critchley LA. Accuracy and precision of the USCOM: does a meta-analysis provide the answer? *Anaesthesia* 2013;68:431–2.
53. Keren H, Burkhoff D, Squara P. Evaluation of a noninvasive continuous cardiac output monitoring system based on thoracic bioimpedance. *Am J Physiol Heart Circ Physiol* 2007;293:H583–9.
54. Raval NY, Squara P, Cleman M, et al. Multicenter evaluation of noninvasive cardiac output measurement by bioimpedance technique. *J Clin Monit Comput* 2008;22:113–9.
55. Squara P, Denjean D, Estagnasie P, et al. Noninvasive cardiac output monitoring (NICOM): a clinical validation. *Intensive Care Med* 2007;33:1191–4.
56. Benomar B, Ouattara A, Estagnasie P, et al. Fluid responsiveness predicted by noninvasive bioimpedance-based passive leg raise test. *Intensive Care Med* 2010;36:1875–81.